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Sleep architecture is related to birth season in 1-month-old infants

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ABSTRACT

Individual variation in sleep quality, quantity, and architecture is pronounced in small infants. Reasons for this remain largely unclear, even though environmental and genetic factors have been suggested to play a role. In order to study the effect of birth seasons on infant sleep architecture, 85 healthy 1-month-old infants underwent an overnight polysomnography (PSG). The PSGs were conducted in 2011–2013. The cohort was divided into four subgroups according to the amount of seasonal light at the time of birth, with each group covering a period of approximately three months. The groups were labeled IL (increasing light), L (light), ID (increasing darkness), and D (dark), corresponding to spring, summer, autumn, and winter, respectively. We found the amount of stage R sleep (precursor of REM sleep, formerly active sleep) to be the highest in infants born in summer, whereas infants born in winter presented the smallest amount of stage R sleep. Infants born in summer presented the smallest amount of stage T sleep (transitional sleep), while stage T sleep was most abundant in infants born in winter. In addition, infants born in summer showed the shortest total sleep time (TST) and the smallest number of awakenings during the study night. This was the first PSG study to find out that birth season modifies the sleep architecture of infants.

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Introduction

Full-term neonates spend approximately over 14 h a day sleeping (Galland et al. 2011, Iglowstein et al. 2003). Daytime sleep and total sleep time decrease gradually as the infant grows, reflecting the maturation of the sleep-wake rhythm and a diminished need for sleep (Galland et al. 2011; Iglowstein et al. 2003).

In the early months of life, sleep comprises three different stages: stage N (precursor of NREM sleep, formerly quiet sleep), stage R (precursor of REM sleep, formerly active sleep), and stage T (transitional sleep) (Berry et al. 2017). Transitional sleep is found especially in wake-to-R-transitions and with arousals, and may cover up to 40% of sleep time in young infants; however, it diminishes with age (Grigg-Damberger 2016).

There is, however, substantial individual variation in the quantity and structure of nocturnal

sleep in infants (Iglowstein et al. 2003; Satomaa et al. 2016). The reasons for this variation are still largely unknown, even though both genes and environmental factors have been suggested to explain it (Fisher et al. 2012; Sadeh et al. 2010; Touchette et al. 2013). The development of diurnal rhythms may also play a role.

Infants start producing melatonin in a diurnal manner as early as at the age of 4–6 weeks (Ardura et al. 2003; McGraw et al. 1999). The circadian clock is entrained via environmental factors (Zeitgebers), the most powerful of which is light. Light-dark cycles are crucial to the development of the circadian sleep-wake rhythm, and the regularity of light-off times helps in entraining the inner clock as early as at the age of 1 month (Iwata et al. 2017). In addition, daily routines have been found to enhance the development of the circadian sleep-wake rhythm in infants (McGraw et al. 1999; Thomas et al. 2016).

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It has been well established that circadian rhythms start developing during the fetal period and continue developing afterbirth. This includes the diurnal secretion of different hormones and the development of the sleep-wake cycle (Mirmiran et al. 2003; Rivkees 2003). The hormonal rhythms become quite well established by around 3–4 months of age (Peirano et al. 2003; Rivkees 2003). However, extensive heterogeneity in postnatal development of both circadian rhythmicity and the sleep-wake cycle has been observed (Galland et al. 2011; Mirmiran et al. 2003; Sadeh et al. 2009), calling for more studies about the moderating factors of infant sleep.

In young infants, the season of the year may have an impact on the secretion of melatonin. For example, in a study conducted on 2-month-olds in Israel, the melatonin production was found to be the highest in infants born in June and lowest in infants born in December; however, the seasonal variations were no longer present two months later (Sivan et al. 2001). According to an actigraphy study by Cohen and co-workers, the sleep onset of infants aged 7 months took place later during the summer months, and the amount of active sleep was higher in summer compared to winter months (Cohen et al. 2012). Therefore, the seasonal amount of light may affect melatonin secretion and sleep development in infants.

The photoperiodic circumstances in northern latitudes are extraordinary and heavily depend on the season, with the daily amount of light varying from just a few hours to approximately 20 h. Knowing the fact that light plays a major role in sleep development, we assume that infants born close to polar regions get a very different basis for their sleep development depending on their season of birth. Our aim is to assess the sleep architecture of infants living in the Tampere region in Finland based on polysomnography (PSG) and then compare the results with the birth season of the infants. We hypothesize that infants born during the dark seasons will have more immature sleep architecture than children born during the seasons of light.

Materials and methods

Our work is part of a larger study (CHILD-SLEEP). CHILD-SLEEP is a multidisciplinary project aiming to evaluate various aspects of sleep in infancy and

early childhood. The study process has been described previously in detail (Paavonen et al. 2017; Satomaa et al. 2016). The present study is conducted on a subgroup of this cohort, comprising infants who underwent an ambulatory overnight PSG at the age of 1 month.

In short, the families were asked to participate in the larger study prenatally at their local maternity clinics. Postnatally, all families who fulfilled the inclusion and exclusion criteria were requested at the maternity ward to participate in the PSG substudy. The inclusion criteria for the PSG study were: healthy, full-term, and uneventful birth (conceptional age 38 weeks or more), Apgar score ≥ 8 at 1 min and birth weight ≥ 2500 g. In addition, the mothers had to be medication-free (from drugs acting on the central nervous system) during their pregnancy. Ultimately, 88 infants born at the conceptional age of 38–42 weeks participated in the PSG study at the age of 1 month (range 3.1–7.9 weeks). The PSGs were conducted in the families' homes in the Tampere region in 2011–2013. The same PSG technician with medical physicists acquired all recordings. The parents were asked to take notes of any events (e.g. feeding and diaper changing) during the night. The study has been approved by the local ethical committees. All the participating parents gave their written informed consent. The birth weight was collected from maternity clinics' database and breastfeeding data was collected using parental questionnaires that were sent to the families at 3 months of age. In addition, the weight was controlled in maternity clinics between 12 and 42 days of age.

Recordings and visual analysis

From the 88 recordings of 1-month-old infants, three were lost because of a technical failure. The ambulatory PSGs were obtained using the Embla Titanium system. The following signals were recorded: six channels of electroencephalography (EEG) (F4-A1, C4-A1, O2-A1, F3-A2, C3-A2, O1-A2), right and left electro-oculography, submental electromyography (EMG), oxygen saturation (pulse oximeter, Nonin), thoracoabdominal inductance plethysmography, diaphragmatic and abdominal EMG, Emfit mattress sensor, and electrocardiography (ECG). Airflow was measured by an oronasal

thermistor (Dymedix). To minimize the sleep disturbance effect of the recording equipment, the nasal pressure transducer was omitted from the protocol (Goodwin et al. 2001). The PSGs were scored into sleep stages in 30-s epochs with the Somnologica Studio 5.0 software by two independent, experienced clinical neurophysiologists. The inter-rater agreement of the scorers was 80.6% (kappa score 0.73 indicating substantial agreement). The scoring differences were discussed, and a consensus scoring was established for further analysis. Because the recordings were started in 2011 when the newest infant sleep staging rules (Berry et al. 2017) had not yet been introduced, the sleep stages were scored according to the AASM 2012 scoring manual (Berry et al. 2012a) with some modifications as detailed in our previous work (Satomaa et al. 2016).

In short, the EEG was scored as stage R, stage N, transitional sleep (T) or wakefulness (W). The R, N, and W were scored according to the REM sleep (R), wakefulness (W), and deep sleep (N3) rules of the AASM 2012 manual, respectively. In addition, stage N was scored when the EEG presented either (1) a high voltage slow pattern on any EEG channel for at least 20% of an epoch's duration or (2) a tracé alternant pattern. To support the decision-making, irregular breathing patterns, and variable heart rates were considered to favor stage R. Stage T was scored when an epoch could not be scored as stage R, N or W. That way, the rules used were practically the same as the new scoring recommendations for 0–2-month-old children (Berry et al. 2017), but since the recordings were ambulatory home recordings, a video was not registered. The duration of each sleep state divided by total sleep time (TST) was expressed as R%, N%, and T%.

The short cortical arousals were scored according to the current guidelines (Berry et al. 2012b; Grigg-Damberger et al. 2007; The International Pediatric Work Group on Arousals (IPWG), 2005). An arousal with a duration of ≥ 30 s, or an arousal followed by a wake epoch was equaled to an awakening. The numbers of awakenings and arousals divided by TST were calculated as an awakening index (AWI) and arousal index (ARI), respectively. In addition, these indexes were summed together to form a combined awakening and arousal index (AWARI).

Statistical analysis

The city of Tampere is located in southern Finland (latitude 61.5°). In the Tampere region, the daily amount of light varies significantly, from a minimum of 5.3 h during the darkest winter period to a maximum of 19.5 h over the lightest summertime. Cut-off points for seasonal light were adjusted according to a yearly sun graph for the Tampere region (Time and date AS, 2018). The cut-off points were set to the beginning of February, May, August, and November. They allowed us to separate four equally long periods with a daily light variation of approximately 5–19 h: the period of dark (group D, Nov 7–Feb 4) with 5.3–8 h of daily light, the period of increasing light (group IL, Feb 5–May 5) with 8–16.5 h of daily light, the period of light (group L, May 6–Aug 5) with 16.5–19.5 h of daily light, and the period of increasing darkness (group ID, Aug 6–Nov 6) with 16.75–8 h of daily light.

The infant sleep data obtained by the PSG were analyzed according to the four birth seasons defined above. The conceptional age of infants at the time of the PSG varied from 41.3 weeks to 48.4 weeks. This may have had an impact on the individual seasons of the PSG recordings. Therefore, TST, T% and R% were analyzed according to the PSG recording season, respectively. Breastfeeding at 3 months of age was categorized into two classes: infants being breastfed only vs. infants having both breast milk and infant formula, or infant formula only.

The associations of birth season, age, birth weight, breastfeeding, and sex were first studied using pairwise statistical tests. Normality of data was assessed by visual inspection. In birth season groups, the Non-Gaussian variables were percentage stage N sleep (N%), number of awakenings, and AWI, while other variables were normally distributed (i.e. Gaussian). In the PSG recording season groups, the TST, T%, and R% were normally distributed.

Gaussian variables were analyzed using one-way analysis of variance while Non-Gaussian variables and variables with different variances were analyzed with the Kruskal–Wallis test. Post-hoc tests were performed using Tukey's test and Dunn–Bonferroni's test, respectively. Categorical variables were analyzed using the Chi-square test.

Linear mixed effects models were computed to study the effect of season of birth while controlling first for sex, and age, and the also for breastfeeding and birth weight. All sleep variables were assessed in separate models. Finally, the interactions between birth season and age were tested and all statistically significant interactions were reported. The differences between T%, R%, and N% in the four birth season groups were analyzed using the Friedman test, with the post-hoc test being Wilcoxon. Statistical analyses were performed using SPSS version 22.

Results

Comparisons between the birth season groups

Among the 85 infants registered at 1 month of age, there were 13 (15.2%) infants born during spring (Feb 5–May 5, increasing light, group IL), 15 (17.6%) born during summer (May 6–Aug 5, light, group L), 41 (48.2%) born during autumn (Aug 6–Nov 6, increasing darkness, group ID), and 16 (18.8%) born during winter (Nov 7–Feb 4, dark, group D). There was no significant difference in sex or conceptional age of the infants in the four birth season groups (p -values 0.580 and 0.800, respectively). The start time of registration (before 21:00 or after 21:00) did not vary significantly within the groups either ($p = .885$). The sleep parameters of the entire cohort are presented in our previous work (Satomaa et al. 2016).

The mean birth weight in the whole cohort was 3571.1 g, with minimum being 2645 g and maximum 4580 g. Two infants were missing birth weight data. Vast majority ($N = 64$, 75.3%) of infants were still breastfed at 3 months of age. Thirteen of infants (15.3%) had infant formula in addition to being breastfed and five infants (5.9%) were having infant formula only, with three (3.5%) infants missing breastfeeding data. There was no significant difference in birth weight or breastfeeding frequency at 3 months of age in four birth season groups ($p = .171$ and $p = .304$, respectively). The control weight between 12 and 42 days of age did not differ between the groups ($p = .996$).

The mean sleep parameters of the four birth season groups are displayed in Table 1. TST differed significantly among the four groups ($p = .026$). The post-hoc

test indicated that TST was longer in group ID than in group L ($p = .018$). T% differed among the groups ($p = .001$) and was lowest in group L. The post-hoc test revealed that T% was lower in group L than in groups ID and D (p -values 0.008 and 0.003, respectively). R% differed among the groups ($p = .020$), and in the post-hoc test comparison, R% was higher in group L than in group D ($p = .015$). The number of awakenings differed among the groups ($p = .047$), being smaller in group L than in group D ($p = .044$). There was no statistical difference in time in bed (TIB) between the groups ($p = .341$).

Comparisons between the PSG recording season groups

In order to evaluate the possible effect of the PSG recording time, we analyzed TST, T%, and R% according to the PSG recording season, respectively. TST differed among the groups ($p = .050$), and the post-hoc test indicated that TST was longer in group D than in group IL ($p = .027$). T% differed among the groups ($p < .001$) and was lowest in group L. The post-hoc test revealed that T% was lower in group L than in groups IL, ID, and D (p -values 0.001, 0.003 and <0.001 , respectively). R% differed among the groups ($p < .001$), being the highest in group L. In the post-hoc test comparison, R% was higher in group L than in groups IL and D ($p = .005$ and <0.001 , respectively), and R% was higher in group ID than in group D ($p = .043$).

Sleep stages within the groups

N% was more abundant than T% in all four birth season groups (p -values ≤ 0.001). The amount of R was higher than T in all groups (p -values ≤ 0.003). In groups ID and D, however, the infants presented significantly less R% than N% ($p = .019$ and <0.001 , respectively), whereas no statistically significant differences between R% and N% were observed in the other groups. These findings are illustrated in Figure 1.

Linear mixed models

In the linear-mixed models, we controlled the results regarding birth season for age and sex. In these analyses, the main results regarding TST and

Table 1. Sleep parameters of 1-month-old infants (n = 85) according to birth seasons.

Parameter ¹	Type ²	Group (Birth season)						Adjusted p-value			
		Increasing light (IL) (5. Feb-5. May) (n = 13)		Light (L) (6. May-8. Aug) (n = 15)		Increasing darkness (ID) (6. Aug- 6. Nov) (n = 41)			Dark (D) (7. Nov - 4. Feb) (n = 16)		
		Average ³	Variation ⁴	Average ³	Variation ⁴	Average ³	Variation ⁴		Average ³	Variation ⁴	p-Value ⁵
TIB (min)	G	731.9	78.4	684.0	88.5	725.5	84.1	722.5	67.6	ns	ns
TST (min)	G	464.3	67.4	439.0	66.4	494.1	59.9	474.4	50.7	0.026	0.032
T%	G	19.5	4.5	17.4	4.5	22.7	6.2	24.3	4.5	0.001	< 0.001
N%	N	42.6	40.0–45.2	41.5	38.7–44.1	40.3	36.1–43.8	41.2	40.3–45.2	ns	ns
R%	G	38.3	7.2	40.6	5.7	36.3	7.5	33.3	4.3	0.020	0.010
SEI%	G	63.7	8.7	64.4	7.6	68.3	6.1	65.8	5.7	ns	ns
WAKE (min)	G	245.0	81.5	234.8	73.5	218.0	61.4	233.6	44.2	ns	ns
No of awak	N	72.0	57.5–76.0	57.0	50.0–66.0	64.0	55.5–80.5	74.0	58.8–86.5	0.047	ns
AWI	N	8.7	7.8–10.8	7.3	6.3–10.3	8.1	6.7–9.3	9.2	7.7–10.7	ns	ns
No of arous	G	79.7	27.2	75.5	15.5	91.5	28.5	83.6	23.6	ns	ns
ARI	G	10.2	3.1	10.4	2.1	11.1	3.3	10.5	2.5	ns	ns
AWARI	G	19.3	3.8	18.6	3.0	19.4	4.1	19.6	3.3	ns	ns
Concage	G	44.4	1.9	44.5	1.3	44.7	1.2	44.8	1.3	ns	ns
G/B (n)	N	9/4		9/6		19/22		6/10		ns	ns

¹TIB = Time in bed. TST = Total sleep time. T% = Percentage of transitional sleep of TST. N % = Percentage of stage N sleep of TST. R % = Percentage of stage R sleep of TST. SEI % = Sleep efficiency index (TST/TIB x 100). WAKE = time spent awake during psg (minutes). No of awake (n) = Number of awakenings per night. AWI = awakening index per hour. No of arous (n) = Number of arousals per night. ARI = arousal index per hour. AWARI = sum of awakenings and arousals per hour. Concage = conceptual age during registration. G = number of girls in the groups. B = number of boys in the groups.

²G = Gaussian, N = Non-Gaussian.

³With gaussian variables, mean is presented, with non-gaussian variables median. Sex is presented in frequencies.

⁴With gaussian variables, SD is represented, with non-gaussian variables quartiles.

⁵With gaussian variables, one-way Anova was used, with non-gaussian variables Kruskal–Wallis test. With categorical variables, Chi–Square was used.

Post-hoc tests showed significant differences between groups as follows: TST: L vs. ID (p = 0.018); T%: L vs. ID (p = 0.008) and L vs. D (p = 0.003); R%: L vs. D (p = 0.015); no of awake: L vs. D (p = 0.044)

sleep stages T and R remained, with the p -values of 0.032 for TST, ≤ 0.001 for T%, and 0.010 for R%. In addition, TST was dependent on conceptional age ($p = .016$). None of these variables were dependent on sex. When controlled further with aforementioned variables, birth weight and breastfeeding at 3 months of age all the main results (TST, T %, R %) remained. There were no significant interactions between conceptional age, sex, and birth season regarding any of the sleep variables we studied.

Discussion

This work is the first PSG study concentrating on birth seasonal differences in sleep stages of small infants. The most important findings of our study were that children born during the season of light (group L) had less transitional sleep (T%) and more R sleep (R%) than the children born during the season of dark (group D). The amount of N sleep did not differ among the four birth season groups. The findings highlight the effect of birth season on infant sleep architecture. Main results remained when analyzed both according to the PSG recording season and controlled for sex, conceptional age, birth weight, and breastfeeding.

In general, during the first year of an infant's life, the daily proportion of TST and the amount of T sleep and R sleep decreases, while N sleep and wakefulness increase (Mirmiran et al. 2003). In healthy infants carried to term, the amount of T is found to be 10–40% of TST (Grigg-Damberger 2016). It contains physiological features of both R sleep and N sleep (Berry et al. 2017). T sleep diminishes during infancy (Ficca et al. 2000), disappearing by 6 months of age (DE WEERD and VAN DEN BOSSCHE 2003). It has been stated that the reduction of T may reflect functional maturation of the central nervous system (Ficca et al. 2000). Since the amount of T sleep was minor in group L, our findings indicate sleep architecture being possibly more mature in infants born during summer.

The other finding supporting the view that summertime is beneficial to infant sleep is that the number of awakenings tended to be lower in group L. It is possible that the natural light circumstances during

a Finnish summer are beneficial to sleep quality, whereas during winter there is substantially less natural light but usually more artificial light, which may cause sleep disturbances (Cho et al. 2015). Indeed, according to a previous study, babies who slept well at night were exposed to significantly more light in the afternoon (12:01–16:00) (Harrison 2004). Similarly, the paucity of awakenings and T sleep might reflect the same phenomenon: stage T sleep occurs by definition more during sleep stage transitions, so it is possible that the reduction of T% in group L is due to fewer awakenings and more stable sleep during summertime.

In addition, in our study, group L had more R sleep than group D. In the analysis of sleep stages within the groups, there was significantly less R sleep than N sleep in group D. Normally R sleep is abundant both in preterm and term infants, diminishing to a level of 30% at the age of 6 months (DE WEERD and VAN DEN BOSSCHE 2003). In our cohort, the amount of R% was 40.6% in group L and 33.3% in group D. Lighting in one potential factor behind these findings, too, as follows.

Children born in northern latitudes during summertime are exposed to considerably more natural light over a period of 24 h when compared with wintertime. In the Tampere region (latitude 61.5°), the maximum daily light time is 19.5 h vs a minimum of less than 6 h. Thus, it is possible that the lighting plays a role in the differences in sleep architecture during different seasons. This is supported by one Mediterranean actigraphy study conducted on 7-month-old infants. The study found that the amount of active sleep (stage R) was higher during summer months than during winter months (Cohen et al. 2012). It can be speculated whether the abundance of R% and paucity of T% in group L in our study is due to the effect of daytime or nocturnal light on increasing R, or decreasing T.

Both stage R sleep development and R reduction are temporally connected to brain development and may be considered as a measure of brain maturation (Mirmiran et al. 2003). It is unlikely that differences in age-related maturation of the infants would affect the season-dependent variation of R and T since there was no difference in conceptional ages among the four birth season groups. In a recent review, Dereymaeker and co-workers estimated the amount

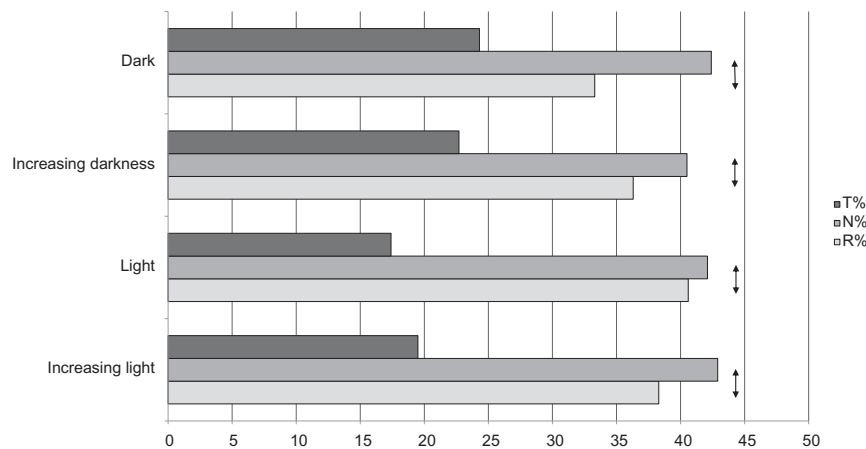


Figure 1. Means of sleep stage percentages R, N, and T in seasonal subgroups.

All differences between the sleep stage percentages were statistically significant ($p = <0.001$ – 0.019), except the difference between N% and R% in the increasing light and light group. Instead in the groups of increasing darkness and dark, N% was significantly higher than R%. Differences between N% and R% are presented with arrows.

of active/R sleep being as high as 45% of TST in 1-month-old infants (Dereymaeker et al. 2017). In one study, a larger amount of REM sleep in premature infants was associated to a better cognitive outcome at 6 months of age (Arditi-Babchuk et al. 2009). This raises the question of whether the early reduction of R sleep in group D might not be beneficial. However, more research is needed to clarify the meaning of this finding.

Light plays an important role in melatonin secretion, which is at its largest during the hours of darkness and may be impacted by artificial illumination (Burgess and Molina 2014; Gooley et al. 2011; Higuchi et al. 2014). Melatonin secretion cyclicality is found to be present as early as around the age of 1 month (Ardura et al. 2003; McGraw et al. 1999). There seems to be seasonal variation in melatonin secretion as well: in a Finnish study, higher levels of nighttime melatonin were observed during wintertime than during summertime in healthy young adults (Pääkkönen et al. 2008). This is expected to shorten the TST in summertime. Indeed, in our study, infants in group L slept less than the infants in groups ID and D. However, according to a study conducted in Israel, nocturnal melatonin secretion was higher in small infants born during the summer months compared with infants born during winter, but the finding did not remain two months later. The result suggests that the regulation of melatonin production could be different in young infants as opposed to later, after maturation. (Sivan et al.

2001) An Australian study did not find seasonal differences in melatonin rhythmicity in 12-week-old infants; however, they found a peak in melatonin excretion in the spring (Kennaway et al. 1996).

The association between melatonin levels and seasonal changes is one potential explanatory factor behind our finding of R% increment in group L, since previous studies have found exogenous melatonin to augment REM sleep in adults (Cajochen et al. 1997; Dijk and Cajochen 1997; Kunz et al. 2004). It is possible that small infants are more susceptible to seasonal changes in melatonin secretion, but the previous findings remain somewhat controversial and the significance of seasonal melatonin secretion differences on infants sleep architecture is still unresolved. Artificial light during winter days and evenings may play a role in some of these findings by suppressing melatonin secretion in newborns.

It is also possible that the excessive amount of light during summer nights explains the abundance of R in group L, since according to Cho and co-workers, artificial light during the night increased the amount of REM sleep in adults (Cho et al. 2016, 2018). Similar findings have been observed in a PSG study of adults in extreme nocturnal light conditions in the Antarctic summer, with REM sleep abundance and deep sleep decrement being observed (Pattyn et al. 2017).

During dark winter months, the need for artificial lighting is substantial compared with

summertime. Artificial light during daytime and evenings may have played a role in suppressing the amount of R sleep in group D, because on the other hand, higher maximal light intensity during daytime has resulted in a lower percentage of REM sleep during subsequent night sleep in healthy adults (Wams et al. 2017). Children seem to be more sensitive than adults in suppressing melatonin production during nighttime due to nocturnal light exposure (Higuchi et al. 2014), but since the previous findings concerning light exposure, melatonin and R sleep are controversial to our findings, further studies are needed.

There have been studies raising the concern regarding artificial and abnormal or continuous lighting on different health issues. Several studies have assessed the potential harmful effects of continuous light environment in neonatal intensive care units (NICU) (Brooks and Canal 2013; Mirmiran and Ariagno 2000; Morag and Ohlsson 2016). According to our findings, light has an effect on infant sleep architecture even in their homes. Further studies are to be needed to assess the effects of artificial light on infant sleep at home.

Our study does have some limitations. The families volunteered to participate in the study, and thus it is possible that they may not accurately represent the Finnish population. Although our sample was randomly recruited, most of the infants in our cohort were born in the autumn and the sample size was smaller in the other birth season groups. We lack information regarding the light circumstances of the infants' bedroom during the PSG night, which is unfortunate knowing that lighting during the night can modify melatonin secretion and sleep architecture (Cho et al. 2016; Gooley et al. 2011; Higuchi et al. 2014). In addition, we do not have information on the family sleep routines and regularity of bedtimes. It is known that family routines and regular light-off times have an impact on the development of sleep rhythm in infants (Iwata et al. 2017; McGraw et al. 1999). On the other hand, there was no difference in the start times of the PSG recording between the four birth season groups. Time spent in bed during the registration (TIB) did not differ among the birth season groups either, even if TIB is found to be the shortest in the spring according to one actigraphy study conducted on adults (O'connell et al. 2014). The stable registration times and TIB may

reflect the families' regular bedtimes independent of the season, so it is unlikely that any deviations in them would explain our results. In this study, however, the exceptions in sleep circumstances due to the PSG recording itself might have had an influence on the sleep quality and quantity of the infants.

Unfortunately, we lack the information on breastfeeding at the time of PSG recording, but we were able to get this information at 3 months of age, when most of the infants were still breastfed. It is highly likely, that those infants were breastfed at PSG timepoint, too. Birth weight data were obtained from vast majority of infants. Weight was controlled in maternity clinics in variable timepoints (12–42 days of age), and therefore this data were not corresponding to PSG recording timepoint and was not suitable for analysis.

When we conducted our sleep EEG analysis, the newest 2017 AASM criteria for infants aged less than 2 months had not been published, but the rules used were practically the same as the new AASM scoring recommendations for 0–2-month-old children (Berry et al. 2017). The sleep stage analyses were carried out by experienced clinical neurophysiologists, using the adapted AASM scoring criteria described elsewhere (Satomaa et al. 2016). Since the recordings were ambulatory home recordings, a video was not registered, which may have made the differentiation of stages R and W more difficult in all the birth season groups. However, notes made by parents were beneficial in differentiating sleep stage R and wakefulness during the study night.

This was the first study to demonstrate that birth season is one potential factor modifying the sleep architecture in small infants. Stage R sleep was abundant during summer and scant during winter, but the clinical significance of this finding remains unclear. To the best of our knowledge, this may reflect the effect of adequate daytime or excessive nighttime light on sleep architecture during summer. Artificial light during dark winter days and evenings is one potential explanatory factor, as well.

Understanding the environmental factors that affect infant sleep quality will help in developing more effective preventive interventions to support or reinforce good sleeping practices and to decrease the risk for developing sleeping difficulties in early childhood. So far, most such interventions are based on improving sleep hygiene and parental practices

around bedtime, while less emphasis is given on the environmental factors, such as seasonal light, that may play a role in sleep quality and quantity.

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Disclosure of interest

The authors report no conflict of interest.

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References

- Arditi-Babchuk H, Feldman R, Eidelman AI. 2009. Rapid eye movement (REM) in premature neonates and developmental outcome at 6 months. *Infant Behav Dev.* 32 (1):27–32.
- Ardura J, Gutierrez R, Andres J, Agapito T. 2003. Emergence and evolution of the circadian rhythm of melatonin in children. *Horm Res.* 59(2):66–72.
- Berry RB, Brooks R, Gamaldo CE, Harding SM, Lloyd RM, Quan SF, Troester MT, VAUGHN BV for the American Academy of Sleep Medicine. (2017). The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Darien (IL): American Academy of Sleep Medicine. Version 2.4.
- VAUGHN BV for the American Academy of Sleep Medicine; Berry RB, Brooks R, Gamaldo CE, Harding SM, Marcus CL. 2012a. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications, Version 2.0. www.aasmnet.org. Darien (Illinois): American Academy of Sleep medicine.
- Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, Marcus CL, Mehra R, Parthasarathy S, Quan SF, et al., AMERICAN ACADEMY OF SLEEP MEDICINE. (2012b). Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events. Deliberations of the sleep apnea definitions task force of the american academy of sleep medicine. *J Clin Sleep Med.* 8(5):597–619.
- Brooks E, Canal MM. 2013. Development of circadian rhythms: role of postnatal light environment. *Neurosci Biobehav Rev.* 37(4):551–560.
- Burgess HJ, Molina TA. 2014. Home lighting before usual bedtime impacts circadian timing: a field study. *Photochem Photobiol.* 90(3):723–726.
- Cajochen C, Krauchi K, Mori D, Graw P, Wirz-Justice A. 1997. Melatonin and S-20098 increase REM sleep and wake-up propensity without modifying NREM sleep homeostasis. *Am J Physiol.* 41(4):R1189–96.
- Cho CH, Lee HJ, Yoon HK, Kang SG, Bok KN, Jung KY, Kim L, Lee EI. 2016. Exposure to dim artificial light at night increases REM sleep and awakenings in humans. *Chronobiol Int.* 33(1):117–123.
- Cho CH, Yoon HK, Kang SG, Kim L, Lee EI, Lee HJ. 2018. Impact of exposure to dim light at night on sleep in female and comparison with male subjects. *Psychiatry Investig.* 15 (5):520–530.
- Cho Y, Ryu S, Lee BR, Kim KH, Lee E, Choi J. 2015. Effects of artificial light at night on human health: A literature review of observational and experimental studies applied to exposure assessment. *Chronobiol Int.* 32(9):1294–1310.
- Cohen D, Atun-Einy O, Scher A. 2012. Seasonal effect on infants' sleep regulation: a preliminary study in a Mediterranean climate. *Chronobiol Int.* 29 (10):1352–1357.
- DE WEERD AW, VAN DEN BOSSCHE RA. 2003. The development of sleep during the first months of life. *Sleep Med Rev.* 7(2):179–191.
- Dereymaeker A, Pillay K, Vervisch J, DE VOS M, VAN HUFFEL S, Jansen K, Naulaers G. 2017. Review of sleep-EEG in preterm and term neonates. *Early Hum Dev.* 113:87–103.
- Dijk DJ, Cajochen C. 1997. Melatonin and the circadian regulation of sleep initiation, consolidation, structure, and the sleep EEG. *J Bio Rhythms.* 12(6):627–635.
- Ficca G, Fagioli I, Salzarulo P. 2000. Sleep organization in the first year of life: developmental trends in the quiet sleep-paradoxical sleep cycle. *J Sleep Res.* 9(1):1–4.
- Fisher A, VAN JAARSVELD CH, Llewellyn CH, Wardle J. 2012. Genetic and environmental influences on infant sleep. *Pediatrics.* 129(6):1091–1096.
- Galland BC, Taylor BJ, Elder DE, Herbison P. 2011. Normal sleep patterns in infants and children: A systematic review of observational studies. *Sleep Med Rev.* 16(3):213–222.
- Goodwin JL, Enright PL, Kaemingk KL, Rosen GM, Morgan WJ, Fregosi RF, Quan SF. 2001. Feasibility of using unattended polysomnography in children for

- research–report of the Tucson Children’s Assessment of Sleep Apnea study (TuCASA). *Sleep*. 24(8):937–944.
- Gooley JJ, Chamberlain K, Smith KA, Khalsa SB, Rajaratnam SM, VAN REEN E, Zeitzer JM, Czeisler CA, Lockley SW. 2011. Exposure to room light before bedtime suppresses melatonin onset and shortens melatonin duration in humans. *J Clin Endocrinol Metab*. 96(3):463–472.
- Grigg-Damberger MM. 2016. The visual scoring of sleep in infants 0 to 2 months of age. *J Clin Sleep Med*. 12(3):429–445.
- Grigg-Damberger MM, Gozal D, Marcus CL, Quan SF, Rosen CL, Chervin RD, Wise M, Picchietti DL, Sheldon SH, Iber C. 2007. The visual scoring of sleep and arousal in infants and children. *J Clin Sleep Med*. 3(2):201–240.
- Harrison Y. 2004. The relationship between daytime exposure to light and night-time sleep in 6–12-week-old infants. *J Sleep Res*. 13(4):345–352.
- Higuchi S, Nagafuchi Y, Lee SI, Harada T. 2014. Influence of light at night on melatonin suppression in children. *J Clin Endocrinol Metab*. 99(9):3298–3303.
- Iglowstein I, Jenni OG, Molinari L, Largo RH. 2003. Sleep duration from infancy to adolescence: reference values and generational trends. *Pediatrics*. 111(2):302–307.
- The International Pediatric Work Group on Arousals (IPWG). 2005. The scoring of arousals in healthy term infants (between the ages of 1 and 6 months). *J Sleep Res*. 14(1):37–41. doi:10.1111/j.1365-2869.2004.00426.x
- Iwata S, Fujita F, Kinoshita M, Unno M, Horinouchi T, Morokuma S, Iwata O. 2017. Dependence of nighttime sleep duration in one-month-old infants on alterations in natural and artificial photoperiod. *Sci Rep*. 7(1):44749.
- Kennaway DJ, Goble FC, Stamp GE. 1996. Factors influencing the development of melatonin rhythmicity in humans. *J Clin Endocrinol Metab*. 81(4):1525–1532.
- Kunz D, Mahlberg R, Müller C, Tilmann A, Bes F. 2004. Melatonin in patients with reduced REM Sleep duration: two randomized controlled trials. *J Clin Endocrinol Metab*. 89(1):128–134.
- Mcgraw K, Hoffmann R, Harker C, Herman JH. 1999. The development of circadian rhythms in a human infant. *Sleep*. 22(3):303–310.
- Mirmiran M, Ariagno RL. 2000. Influence of light in the NICU on the development of circadian rhythms in preterm infants. *Semin Perinatol*. 24(4):247–257.
- Mirmiran M, Maas YG, Ariagno RL. 2003. Development of fetal and neonatal sleep and circadian rhythms. *Sleep Med Rev*. 7(4):321–334.
- Morag I, Ohlsson A. 2016. Cycled light in the intensive care unit for preterm and low birth weight infants. *Cochrane Database Syst Rev*. 10(8):CD006982.
- O’connell SE, Griffiths PL, Clemes SA. 2014. Seasonal variation in physical activity, sedentary behaviour and sleep in a sample of UK adults. *Ann Hum Biol*. 41(1):1–8.
- Pääkkönen T, Leppäluoto J, Mäkinen TM, Rintamäki H, Ruokonen A, Hassi J, Palinkas LA. 2008. Seasonal levels of melatonin, thyroid hormones, mood, and cognition near the arctic circle. *Aviat Space Environ Med*. 79(7):695–699.
- Paavonen EJ, Saarenpää-Heikkilä O, Polkki P, Kylliäinen A, Porkka-Heiskanen T, Paunio T. 2017. Maternal and paternal sleep during pregnancy in the Child-sleep birth cohort. *Sleep Med*. 29:47–56.
- Pattyn N, Mairesse O, Cortoos A, Marcoen N, Neyt X, Meeusen R. 2017. Sleep during an Antarctic summer expedition: new light on “polar insomnia”. *J Appl Physiol*. 122(4):788–794.
- Peirano P, Algarin C, Uauy R. 2003. Sleep-wake states and their regulatory mechanisms throughout early human development. *J Pediatr*. 143(4 Suppl):70–79.
- Rivkees SA. 2003. Developing circadian rhythmicity in infants. *Pediatrics*. 112(2):373–381.
- Sadeh A, Mindell JA, Luedtke K, Wiegand B. 2009. Sleep and sleep ecology in the first 3 years: a web-based study. *J Sleep Res*. 18(1):60–73.
- Sadeh A, Tikotzky L, Scher A. 2010. Parenting and infant sleep. *Sleep Med Rev*. 14(2):89–96.
- Satoma AL, Saarenpää-Heikkilä O, Paavonen EJ, Himanen SL. 2016. The adapted American Academy of sleep medicine sleep scoring criteria in one month old infants: A means to improve comparability? *Clin Neurophysiol*. 127(2):1410–1418.
- Sivan Y, Laudon M, Tauman R, Zisapel N. 2001. Melatonin production in healthy infants: evidence for seasonal variations. *Pediatr Res*. 49(1):63–68.
- Thomas K, Burr R, Spieker S. 2016. Light and maternal influence in the entrainment of activity circadian rhythm in infants 4–12 weeks of age. *Sleep Biol Rhythms*. 14(3):249–255.
- Time and date AS. (2018). Sunrise and sunset times in Tampere. Stavanger (Norway): Time and date AS. Available from: <https://www.timeanddate.com/sun/finland/tampere> [last accessed 16 Dec 2018].
- Touchette E, Dionne G, Forget-Dubois N, Petit D, Perusse D, Falissard B, Tremblay RE, Boivin M, Montplaisir JY. 2013. Genetic and environmental influences on daytime and nighttime sleep duration in early childhood. *Pediatrics*. 131(6):1874–1880.
- Wams EJ, Woelders T, Marring I, VAN ROSMALEN L, Beersma DGM, Gordijn MCM, Hut RA. 2017. Linking light exposure and subsequent sleep: a field polysomnography study in humans. *Sleep*. 40(12). doi:10.1093/sleep/zsx165